

## CLINICAL IMAGE

# Pulmonary oedema after acute upper airway obstruction

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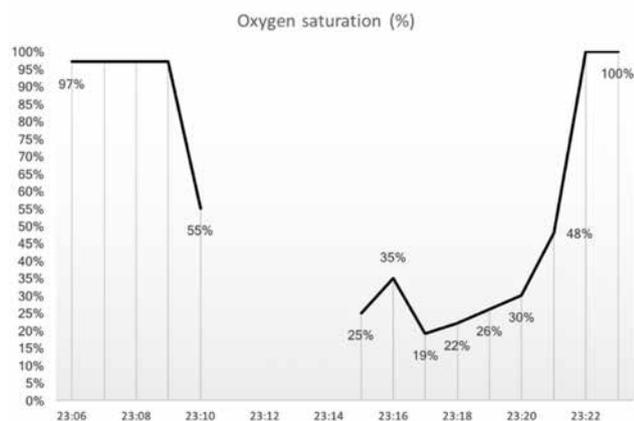
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**Keywords** - pulmonary oedema, acute respiratory failure, epiglottitis, tracheotomy, upper airway obstruction

## Case presentation

A 48-year-old male, with no medical history, was admitted to the emergency room with dyspnoea and a sore throat. The pain started the previous day, but had become worse and he was experiencing dysphagia. He was taking 2000 mg ibuprofen and 2000 mg ibuprofen for these symptoms. Three hours later he started to experience dyspnoea. During physical examination in the emergency room, the patient was distressed, with an inspiratory stridor, slightly swollen lips and a painful erythematous neck.



**Figure 1.** The upper airway obstruction started after 23.09h, between 23:10-23:15h there was no signal of the saturation meter. But for at least, and probably longer, 5 minutes the oxygen saturation stayed below 35%. Rapid normalization of oxygen saturation occurred after placing a tracheostomy cannula

The respiratory sounds were normal on both sides with an oxygen saturation of 98% without additional oxygen. He was haemodynamically stable, Glasgow Coma Scale was 15 points and his temperature was 38.2 °C. Blood tests showed: CRP 54 mg/l, leukocytes 17.6/nl, potassium 3.3 mmol/l and sodium 138 mmol/l. On direct laryngoscopy, the otolaryngologist observed a diffusely erythematous and swollen epiglottitis

which collapsed during inspiration. Vision of the larynx was completely obstructed. Acute epiglottitis was suspected. In the differential diagnosis an allergic reaction to ibuprofen was also considered. He was given 1000/200 mg of amoxicillin/clavulanic acid and 2 mg of clemastine (antihistamine and anticholinergic) intravenously and was admitted to the intensive care unit (ICU) for monitoring. The intensivist and otolaryngologist chose not to sedate and intubate the patient in the emergency room. First because of the stable pulmonary state without stridorous breathing, and second because they did not expect the situation to worsen in the immediate future.



**Figure 2.** One hour after tracheotomy a chest X-ray was performed, showing diffuse pulmonary edema

Thirty minutes after arrival on the ICU the patient experienced an acute obstruction of the upper airway. He was frightened, agitated and rapidly lost consciousness with concurrent deep cyanosis. The otolaryngologist and intensivist performed an emergency cricothyrotomy. Securing an open airway was the most important thing to do at that moment. Because of initial

doubt that the airway was secured and a further decrease in oxygen saturation reaching 19% (*figure 1*), they performed a percutaneous tracheotomy. The airway was secured and the oxygen saturation rose quickly. One hour later an X-ray of the thorax was performed (*figure 2*) which showed pulmonary oedema. The next day the patient awoke with a normal neurological status. During the following days the patient fully recovered, the tracheal cannula was removed and he could be successfully discharged home.

## Discussion

Post-obstructive pulmonary oedema (POPE) or negative-pressure pulmonary oedema (NPPE) usually occurs as a result of upper airway infection, tumour or laryngospasm.<sup>[1]</sup> In 1927, Moore demonstrated a relationship between pulmonary oedema after airway obstruction in a group of dogs.<sup>[2]</sup> In humans two different types of POPE are recognised: after an acute (e.g. epiglottitis, strangulation, post-extubation laryngospasm) or a chronic airway obstruction (e.g. post-tonsillectomy, post-removal of upper airway tumour, choanal stenosis).<sup>[3]</sup> The pathophysiology of NPPE is mostly hydrostatically driven. Negative pressure causes an increased hydrostatic gradient leading to transcapillary flux and alveolar flooding.<sup>[1,4-5]</sup> With the modified Müller's manoeuvre (deep inspiratory effort against a closed glottis) high levels of negative inspiratory pressure with a maximum of -140 cmH<sub>2</sub>O can be reached.<sup>[6]</sup> Negative intrathoracic pressure is transmitted to the intrapleural spaces. This causes an increase in the venous return to the right side of the heart, decreased left ventricular stroke volume and pulmonary venous pressures and the perivascular interstitial hydrostatic pressure is decreased. Subsequently a flux of fluid from pulmonary capillaries into the interstitium and alveolar spaces takes place.<sup>[5,7,8]</sup> In the event of acute upper airway obstruction, the hyperadrenergic state can cause peripheral vasoconstriction and an increase in venous return. This leads to an increase in pulmonary blood flow and concomitant oedema. Additionally, the hydrostatic pulmonary

oedema observed in patients suffering from congestive heart failure is similar to NPPE. There is a second mechanism that potentially could have played a role in our patient, namely the high negative pressure could have induced discontinuities in the alveolar epithelial and pulmonary microvascular membranes leading to protein-rich oedema. However, several studies show a relatively low ratio of protein in the oedema,<sup>[9,10]</sup> suggesting that the hydrostatic driven mechanism for oedema formation may be most prominent in patients with acute upper airway obstruction. In conclusion, NPPE is a well-established secondary effect of acute upper airway obstruction and resolves within 24-48 hours in most cases.

## Disclosure

All authors declare no conflict of interest. No funding or financial support was received.

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